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Unexpected reappearance of ST elevation in the anterior precordial leads shortly after an acute anterior myocardial infarction

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Introduction

Brugada syndrome (BrS) is an inherited congenital electrical abnormality characterized by right precordial ST-segment elevation that predisposes patients with apparent structurally normal hearts to malignant ventricular arrhythmias and sudden cardiac death. The diagnosis of BrS is based on typical ECG characteristics along with symptoms and familial history. Brugada phenocopy (BrP) encompasses a number of clinical entities characterized by ECG patterns that, while identical to true BrS, are elicited by various clinical conditions (i.e., metabolic conditions, mechanical compression, myocardial ischemia, myocardial/pericardial diseases, and miscellaneous). ECG normalizes upon resolution of the underlying condition, personal or family history of arrhythmic syncope or ventricular arrhythmias is strictly absent, provocative tests with sodium channel blockers and genetic screening are negative.

Case report

A 52-year-old male smoker, with no prior medical cardiac history, presented to the emergency department with angina chest pain lasting for 1 h. ECG revealed ST-segment elevation in leads V1, V2, V3 and ST-segment depression in inferior leads (Fig. 1). Blood tests were normal except a slight increased high sensitive troponin I: 35 ng/L (<20 ng/L). A diagnosis of acute anterior myocardial infarction was made. Emergent coronary angiography showed a complete thrombotic occlusion in the mid-left anterior descendent (LAD) coronary artery and a 70% stenosis in the distal segment of the right coronary artery (RCA). Balloon angioplasty of LAD (culprit lesion) with placement of a drug-eluting stent was performed. The evolution of ST segment morphology after percutaneous intervention is shown in Fig. 2. Three days later the patient presented intermittent chest pain and ECG revealed a ≥0.2 mV coved-type ST-segment elevation in the right precordial leads ending with a negative T wave, similar to ECG changes seen in BrS (Fig. 3). Personal or family history of unexplained syncope, sudden cardiac death, or implantable cardiac defibrillator implantation were excluded. Serum electrolytes were normal and the patient was not receiving any medication known to trigger a Brugada ECG pattern. A baseline ECG performed three years before did not show any abnormality. A new coronary angiogram showed a patent stent in the LAD coronary artery and ruled out RCA dissection. The stenosis in the distal segment of the RCA was successfully treated by angioplasty and stent implantation. The ECG normalized spontaneously 3 days following PCI of RCA. The ECG evolution of the ST segment from the acute fase and the differences between ST elevation during acute myocardial infarction and those due to BrP were evident (Fig. 4). We did not attempt a challenge test with a sodium channel blocker in view of the low likelihood of true Brugada syndrome and the inherent risks of the procedure related to the recent myocardial infarction. Genetic testing was not performed, giving the low diagnostic yield. The echocardiogram performed during admission ruled out an aneurysm in the anterior and anteroseptal walls. The ECG changes did not recur on follow up and an ECG Holter performed three months later showed no arrhythmia. On the basis of the clinical picture, the fleca or the ajmaline test has been planned six to nine months after the acute event.

Discussion

The association between BrS, BrP and myocardial ischemia is still poorly understood [1,2]. In the literature, some case reports highlight that Brugada syndrome can be mistaken for acute myocardial infarction but the reverse may also occur [3]. In fact myocardial ischemia may either induce a BrP or, inversely, unmask a true congenital BrS. Few clinical descriptions of BrP observed during acute myocardial ischemia can be found in the literature and are properly summarized in the International Registry and Educational Portal on BrP [4]. Recently Ferrando-Castagnetto et al. [2] reported the case of a woman exhibiting BrP during an acute anterior myocardial infarction, while Alper et al. [5] described for the first time a case of BrP in the inferior leads in the context of an acute inferior myocardial infarction. None of these provide an explanation as to how acute ischemia and Brugada ECG pattern may be related. Although research on cellular basis of true BrS is constantly evolving, physiological mechanisms underlying BrP in acute myocardial ischemia are still unknown. Transient outward potassium flux could modulate ECG manifestations of acute ischemia as well as that of BrS, suggesting both clinical entities could be the result of a similar electrophysiological substrate [2–6]. To the best of our knowledge, no prior clinical descriptions of BrP early after acute myocardial infarction can be found in the

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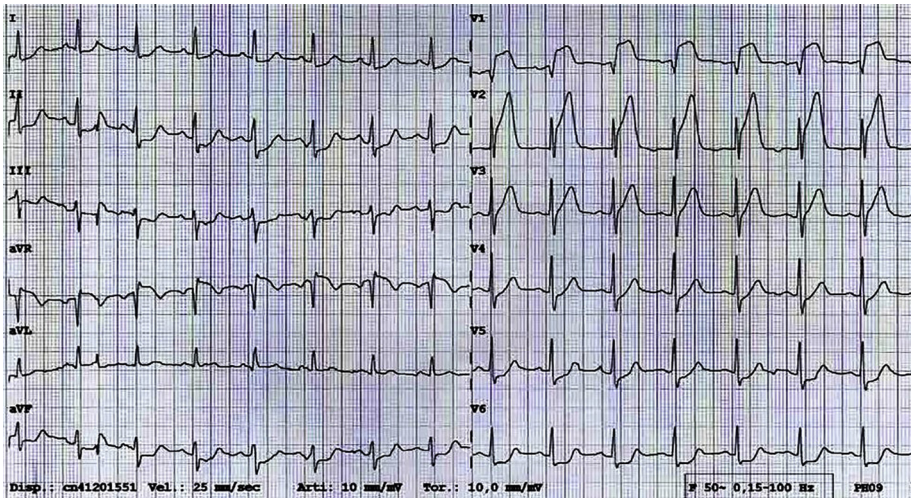

Fig. 1. ECG on arrival in the emergency room showing ST-segment elevation in leads V1, V2, V3 and ST-segment depression in inferior leads.

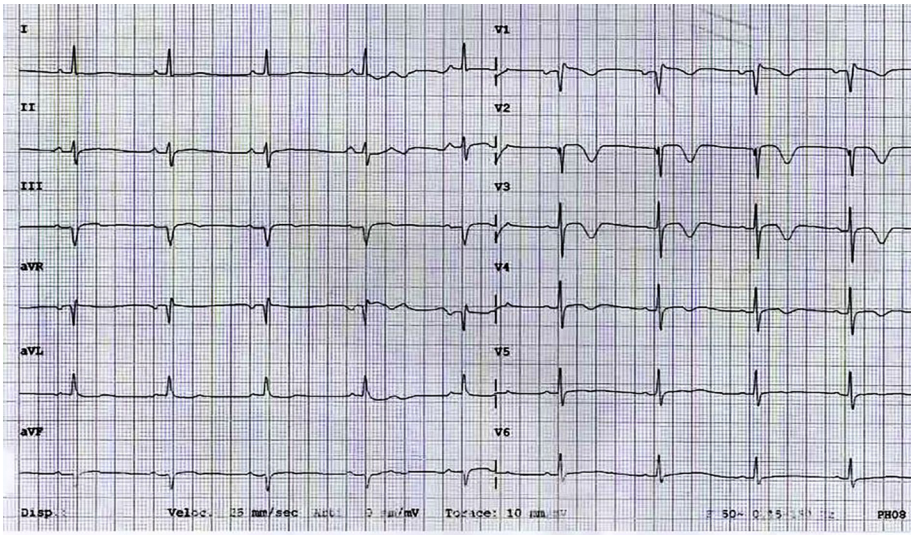

Fig. 2. ECG after balloon angioplasty of LAD and placement of a drug-eluting stent showing ST segment morphology evolution after percutaneous intervention.

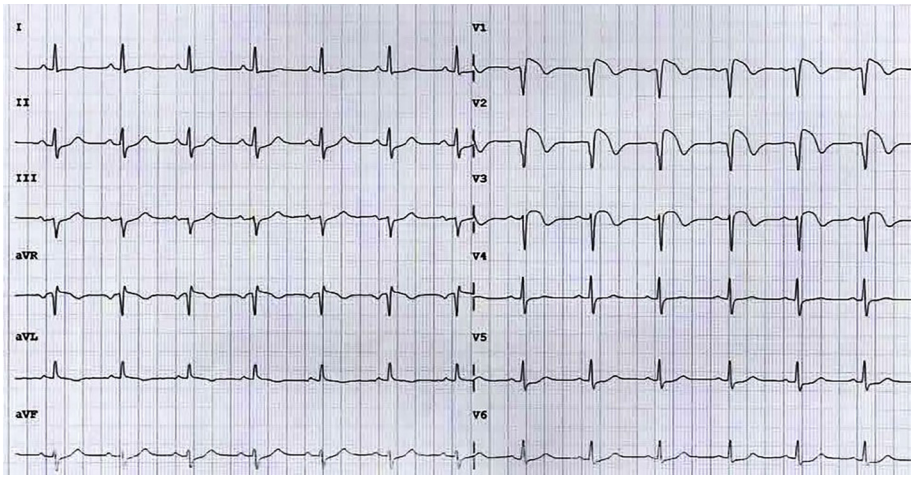

Fig. 3. ECG three days later after balloon angioplasty of LAD showing a ≥0.2 mV coved-type ST-segment elevation in the right precordial leads ending with a negative T wave similar to ECG changes seen in BrS.

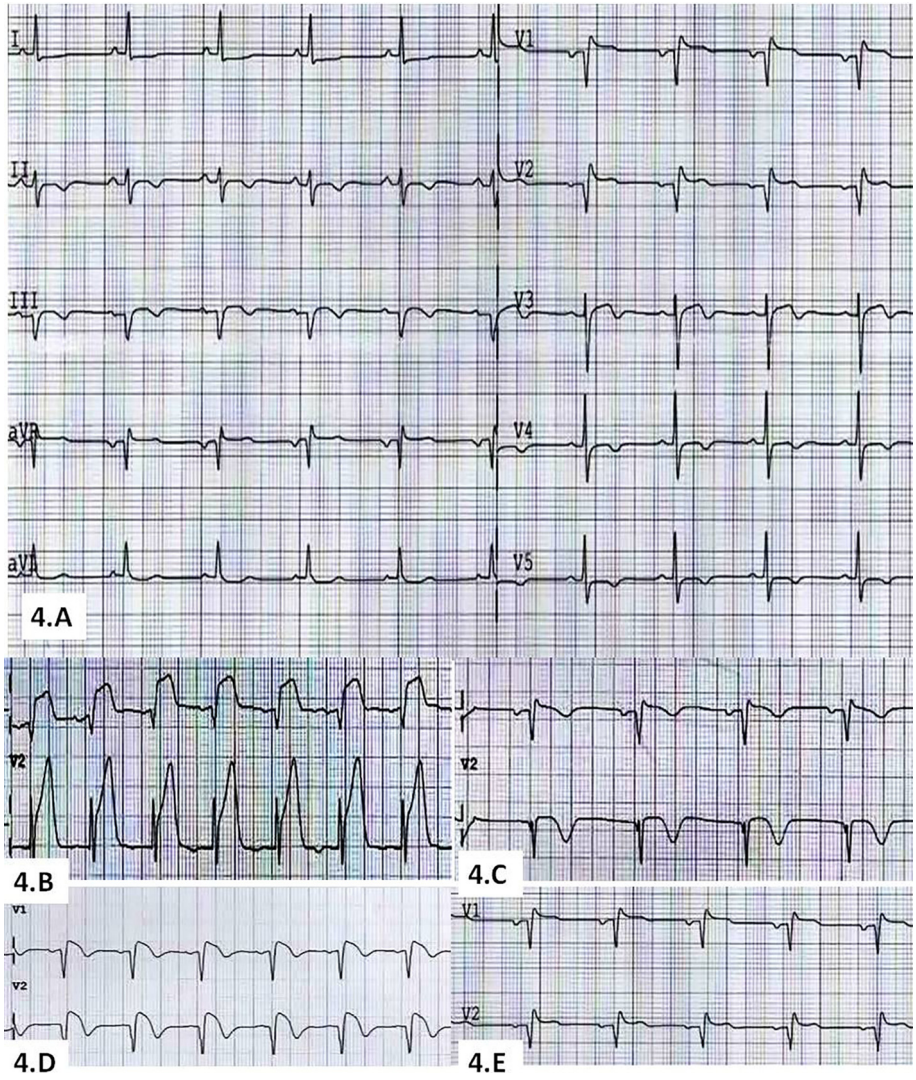

Fig. 4. A: ECG normalization three days later after PCI of CD; B to E: Leads V1–V2 to show ST segment evolution and the difference between ST elevation during STEMI and due to BrP.

literature and are described in the Registry [4–7]. In our case transient ECG coved-type ST-segment elevation in precordial leads V1 and V2 normalized early after complete revascularization and personal or family history of arrhythmic syncope or ventricular arrhythmias was absent. Other possible causes of persistent ST elevation after acute myocardial infarction were excluded. The echocardiogram ruled out an aneurysm in the anterior and anteroseptal walls [8] and the second coronary angiography did not show RCA dissection [9]. Although the case would require a challenge with a sodium channel blocker (ajmaline, flecainide, or procainamide), we did not perform these tests given the severe lifethreatening clinical situation (recent anterior myocardial infarction) and the complete reversal of the ECG pattern early after myocardial complete revascularization. In our patient current diagnostic and morphological criteria proposed by Anselm et al. suggest the diagnosis of Type-1, Class B BrP [7]. The potential mechanisms and pathophysiology underlying Type 1 Brugada ECG pattern remain unclear. The depolarization theory hypothesizes that ST-segment elevation might be caused by the conduction delay in the right ventricular outflow tract [1]. According to this theory, regional right ventricular ischemia induced by RCA stenosis eventually causing potential difference between ischemic and nonischemic regions might be responsible for electric abnormalities eventually leading to ST-segment elevation. The ECG normalization after successful complete revascularization supports this hypothesis. Myocardial ischemia either due to vasospasm or transient embolization of RCA cannot be completely ruled out.

Conclusion

To the best of our knowledge, this represents the first report dealing with a case of type 1 Brugada ECG pattern suggesting Class B BrP early after and not in the context of an acute myocardial infarction. This unusual case may contribute to the growing body of literature on BrP and stimulate further research.

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